and lassitude. The physical signs in her chest were those of fibrosis of the right lung. In July, 1922, signs of cavitation were noticed, the sputum became more profuse, with sweats and irregular temperature, and she died on March 14th, 1924.

An *x*-ray plate showed extensive fibrosis, more marked in the right lung, two calcareous glands at the root of the left lung, and two small calcareous particles in the base of the left lower lobe.

Macroscopical Appearances.

Right Lung.—The pleura is thickened over the entire surface of the lung, and shows the remains of dense adhesions to the chest wall and pericardium. The lung is firm and small. The glands at the root of the lung are large, and on section are black, show a thickened capsule, and some calcareous particles. On section, the lung is seen to be fibrosed and to a large extent airless, the lung tissue being replaced by fibrous tissue. Dense strands of fibrous tissue from the pleura intersect the lung. In the apex there is a large cavity, the size of a peeled tangerine orange. The middle and lower lobes show numerous small areas—varying in size from a hazel-nut to a pin's head—of caseation, some of which have proceeded to cavitation. The bronchi are dilated.

Left Lung.—The pleura is thickened and shows the remains of adhesions to the chest wall. The thickening and adhesions are not so marked as in the right lung. The lung is firmer than normal. At the root of the lung are two large calcareous masses, one the size of a large hazel-nut, the other about half that size—calcified tuberculous glands. The other glands are black and show periadenitis. In the left apex there is an area of old scar tissue about the size of a sixpenny piece, and a cavity the size of a walnut. Scattered throughout the lung are small areas of denser consistence than the rest of the lung, some of which show definite calcareous particles, others small areas of caseation. There is a considerable increase in the fibrous tissue.

Three outstanding features are presented by sections from this case. The first is the enormous amount of fine granular pigment in the peribronchial fibrous tissue, walls of alveoli, and in phagocytes scattered through the sections. The particles of this dust are similar in size and shape to the black granules seen in the asbestos fibre.

The second unusual feature is the presence of large solid angular particles (Fig. 2). These are situated in areas of fibrosis and in caseating areas. They vary in size from 3 to 360 microns in length. The particles are so large—masses of them are seen in certain areas—that they must have occluded small bronchi. Fibrosis of the alveoli supplied has taken place and later necrosis, as seen in Fig. 3.

We have never seen anything parallel to this in pneumonoconiosis due to other dusts, nor have we been able to find such occurrence in literature. On comparing these large particles with asbestos dust there is a striking resemblance in sizes, shapes, and colour. In fact, it is very easy to take each single particle found in the lung sections and immediately find its brother in a slide made from the dust.

We cannot think there is any reasonable doubt that the particles in the lungs are the heavy, brittle, iron-containing fragments of asbestos fibre. The more extensive involvement of the right lung is thus explained. The heavy particles would pass more easily down the more vertical right bronchus than the horizontal left bronchus.

HISTOLOGY OF PULMONARY ASBESTOSIS.

BY

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(*With Special Plate.*)

My remarks are confined to the histological appearances in the lungs in this condition, with special reference to certain foreign bodies of most unusual appearance which are present both in the alveoli and interstitial substance of the lungs. The observations are based almost entirely on material supplied from the case described by Dr. Cooke. The investigation has been conducted in the pathological department of the University of Durham College of Medicine.

I may state, however, that the appearances are practically identical with those observed in a second case of this condition, sections of which I have had an opportunity of examining through the courtesy of Dr. I. M. D. Grieve of Armley, Leeds.

Histology.

Numerous sections have been made from both lungs. The changes are more marked on the right side, but the appearances in the two lungs only differ in degree. They may be summarized as follows:

1. There is well marked diffuse interstitial pneumonia with chronic bronchitis and some emphysema.
2. There is well marked anthracosis.
3. There is an extensive tuberculous condition with chronic phthisis.
4. In the alveoli, bronchi, and bronchioles, and also in the interstitial fibrotic areas, are certain foreign bodies which will be described in detail later (Fig. 7).

As this communication deals specially with the nature of the foreign bodies, the general histological features will be dealt with very briefly.

The interstitial fibrosis is such as might be expected as a result of a combination of a pneumonoconiotic condition and a chronic tuberculous infection. The typical whorled formations seen in a more purely silicotic condition are not present. There is a marked endarteritis in the smaller branches of the pulmonary arteries; some are thrombosed and organized. Many of the smaller bronchi are obliterated; some have still caseous-looking centres. Some of the alveoli show the usual metaplasia of their lining cells into cuboidal form. The fibrosed and thickened walls of the bronchi in many places gradually merge into the areas of diffuse fibrous overgrowth. There are numerous foci of lymphocytic cells among the fibroblasts. Some of these seem obviously derived from lymphoid tissue in the bronchial walls. The interstitial fibrosis is progressive. The tuberculous condition is obvious histologically. Tubercle bacilli were not detected, but the histology is characteristic. The lesions are chronic in character, and there is no special indication of an acute exacerbation. There is well marked caseous bronchitis with lymphatic spread and numerous fibro-caseous deposits with giant cell systems (see Fig. 8). The bronchi, which are not specially the seat of tuberculous change, show catarrh with peribronchial thickening. There are numerous emphysematous areas. The alveoli show, in the majority of cases, some thickening of their walls, and contain many catarrhal cells, apparently derived from the lining cells; a similar catarrhal change is seen in the terminal bronchioles, some of which are dilated.

The Foreign Bodies.

The larger black and irregularly fragmented bodies which have been described by Dr. Cooke were not very obvious in the material I examined, but were clearly seen in some microscopical preparations of his which I had the opportunity of examining. I shall not refer to them specially, but confine my attention to certain highly characteristic and much smaller bodies which are abundant in all the sections examined. Some of these are free, but many are phagocytosed by the large mononuclear cells in the alveoli (Fig. 6). Some are easily included in comparatively small phagocytic cells, but the majority are larger, varying in size from 20 μ to 70 μ, or even more in the case of certain elongated forms. The smaller bodies are rounded and homogeneous, and all have a distinct yellowish-brown colour suggesting blood pigment. The longer forms have a highly characteristic appearance, strongly suggestive of some organic structure. Most have an annular appearance, which on closer examination can be resolved into a closely set series of rounded discoid bodies (Figs. 4, 5, 10, and 11). In some cases the globular forms are arranged along the more filamentous forms and occasionally are clustered at the ends of the rods, simulating sporangia of a hyphomycetes (Figs. 5, 6, 10, and 11). Some have club-like extremities at one or both ends of the filaments. Others, again, suggest the appearance of minute crustacean forms (Fig. 10), but closer examination does not support the idea of either vegetable or animal origin. These bodies do not stain with the ordinary aniline stains, but preserve their original yellow-brown colour. They are seen well in unstained sections. They give a characteristic prussian-blue reaction with potassium ferrocyanide and dilute hydrochloric acid. The reaction is not so obvious unless the solutions are slightly warmed. Where the bodies are too large to be phagocytosed by individual cells they tend to become surrounded by plasmodial masses. Many of the phagocytes contain much carbon pigment in addition. Though these bodies are mainly found in the alveoli and

infundibular passages, they are also present in the caseous areas, in the neighbourhood of the phthisical cavities, and some can be demonstrated in the fibrotic areas surrounded by definite fibroblasts. One in particular (see Fig. 12) measured about 75 μ. It is clear and segmented in its middle part, but the extremities are nodular and clubbed. It is difficult to imagine that a foreign body of such length could be transported by phagocytes, but they may represent larger bodies left in a bronchus which has become obliterated. The bodies have been examined with the micro-spectroscope, but so far no clue as to their nature has been obtained by this means. They are not refractile by polarized light.

Nature of the Bodies.

We have shown these preparations to several pathologists, but the appearances are new to them. To confirm our own opinion we have submitted them to experts in zoology, who are unanimous that they are not of animal nature. We have also submitted them to botanical and chemical authorities, and though there has been a considerable difference of opinion, some regarding them as hyphomycetes, the general opinion has been that they are not vegetable forms.

The fact that exactly similar bodies have been found in the lungs of another asbestos worker, and, so far as I can ascertain, have not been found elsewhere, would seem to indicate that they are essentially derived from or associated in some way with the asbestos itself. It is also certain that they do not in any way resemble concretions, largely composed of calcium and other salts, and also containing iron derived from blood, such as have been described as streptothrix forms in the spleen, and which may closely simulate mycelial filaments. The hypothesis advanced is that these bodies are portions of asbestos fibres in the process of alteration and absorption by hydrolysis, either by direct chemical action or by enzymes. The particular variety of asbestos with which this patient worked was a Canadian serpentine (chrysotile). It would probably contain silica and a magnesium salt in about equal proportions (40 per cent.) with up to 3 per cent. ferrous oxide, 1 per cent. of alumina, and water. From its high resistance to heat we are apt to regard asbestos as indestructible, but, given time, it is possible for hydrolysis of such silicates to occur, even in pure water. Such hydrolysis would be hastened and intensified by the presence of CO_2 in the pulmonary alveoli, and the warm moist atmosphere there would, no doubt, accelerate the process. Even under these conditions the process would necessarily be a slow one. The magnesium could be separated out as relatively insoluble carbonate, or more soluble bicarbonate, which in turn would be converted into any other salt for which there happened to be the appropriate acid available.

The iron existing in a ferrous condition in the presence of an oxidizing agent might be converted into the ferric state, and subsequently precipitated as hydroxide. The silica might pass into a colloidal state, at first in sol form (orthosilicic acid), later passing into a gel (metasilicic acid). If this were so in sol form, it would tend to remain associated with the surface of the asbestos fibre by adsorption, and might be held there till it became a gel. In time the gel might adsorb the solution, and so gradual conversion of the fibre into a mass of gel would occur. There might be in the tissues sufficient albuminoid material to effect rapid gelatinization of the sol, particularly if, as would be the case here, the sol was being slowly produced. The fact that the gel is of high surface tension, and formed at an irregular rate, would give it a spheroidal structure and account for some of the appearances seen here. Whether this be the exact explanation or no, it is at least an hypothesis which should be capable of experimental verification. As has been held by Gye and others, in cases of silicosis there may be a direct chemical action of silica on the tissues apart from the merely mechanical irritation of the particles, with the production of fibrosis. Orthosilicic acid is, as has been shown, an active poison, but rapid conversion into metasilicic acid would minimize its action.

As to the relative part played by the asbestos and the tuberculous infection in this case, in relation to the fibrotic change, it is difficult to say, but it is a reasonable assumption that the tuberculosis was a superadded infection, and in Dr. Grieves's case referred to above there was in the sections examined a considerable degree of fibrosis without apparent tuberculosis. The immediate cause of death in that case was a terminal broncho-pneumonia. Till some experimental work is completed the exact nature of these foreign bodies must remain in doubt, but their highly characteristic appearance may well prove to be an important diagnostic point in the recognition of the lung of a worker in asbestos.

I am much indebted to Dr. Cooke for material from his case, to Dr. Grieve for an opportunity of examining his microscopical sections, to P. L. Robinson, D.Sc., of the Chemical Department, Armstrong College, for his advice and suggestions on the chemistry of the silicates, and to Professor W. H. Lang, F.R.S., of Manchester, for a reasoned opinion as to the non-botanical nature of the foreign bodies.

CLINICAL ASPECTS OF PULMONARY ASBESTOSIS.

BY

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DR. W. E. COOKE has given a short account of the history of asbestos, also of the processes of its manufacture into cloth-like structures much in the same manner as raw cotton fibre is woven. He has told us that in the carding department a considerable quantity of dust is evolved. The crushing of the rock is not carried on to any extent in this country; this is usually done in the countries where the mineral is quarried. Canadian rock is crushed in Canada so as to reduce the expense of transport to Great Britain. Our workers are therefore less exposed to the harmful influence of the dust—a fortunate circumstance, since the rock frequently contains as much as from 50 to 60 per cent. or more of silica.

With the exception of Dr. Cooke's paper on pulmonary asbestosis published in 1924, and the details of a fatal case published by Dr. Montague Murray in the *Charing Cross Hospital Gazette* in 1900, there has not been, to my knowledge, anything written in this country upon the subject. I have had, however, the opportunity of visiting asbestos factories in America, and of seeing cases of pulmonary asbestosis through the kindness of Drs. Haddow and Grieve of Armley, Leeds. It may, I think, be safely said that there must have been several deaths of workers in British factories from the malady, but as no autopsy and microscopical examinations of the lungs were made the deaths were probably certified as pulmonary tuberculosis.

Asbestos manufacture is largely a familial occupation. It has been carried on in this country only for a little over thirty years. Carding and spinning of the fibre are important processes in the manufacture of asbestos goods. In these departments many women are employed, mothers being succeeded by their daughters. Where ventilation of the carding and spinning rooms is properly attended to the atmosphere is fairly clear of dust and floating fibre, otherwise in these operations considerable quantities of dust become suspended in the atmosphere. In a British factory the dustiest process is "hand beating" of the finished mattresses used for covering and protecting the internal machinery of automobiles. This work should only be undertaken in a room separated from the main parts of the factory, with open windows at one end and strong down-draughts at the other, but even with this precaution men working therein should wear masks.

Recently, with Dr. Grieve of Armley, I examined two women who are the subjects of pulmonary asbestosis, one aged 48 and the other 39. The older patient was one of the first to commence work thirty years ago in the particular factory I visited. At that date no danger from dust was anticipated, so that no effective ventilation of the workrooms was attempted, such as prevails to-day. Although only 48 the first patient mentioned looks older by several years, and is extremely emaciated. She gave up work a year ago on account of increasing physical